Kardiologia Polska 2013; 71, 6: 650–652; DOI: 10.5603/KP.2013.0137 ISSN 0022–9032

Acute myocardial infarction in a patient with chronic renal failure and endocarditis

Ostry zawał serca u chorego z przewlekłą niewydolnością nerek i zapaleniem wsierdzia

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Abstract

We report a case of a 55 year-old female with chronic renal failure who received routine haemodialysis and suffered from acute myocardial infarction of inferior wall. Based on coronary angiogram, transoesophageal echocardiography, and autopsy, coronary embolisation with vegetations in the course of infective endocarditis was identified as a rare cause of ST-segment elevation myocardial infarction.

Key words: acute myocardial infarction, endocarditis, chronic renal failure, embolisation, angioplasty

Kardiol Pol 2013; 71, 6: 650–652

Systemic embolisation and, in particular, central nervous system embolisation is a frequent complication of infective endocarditis. Embolised vegetations have been reported also as a rare cause of ST-segment elevation myocardial infarction (STEMI) [1].

A 55 year-old female, with chronic renal failure and arterial hypertension, was admitted to the nephrology unit for routine haemodialysis (she had been receiving haemodialysis for about five years). During dialysis, a sudden drop of blood pressure was noticed and she lost consciousness. ST-segment elevation in II, III, aVF leads was noted in the electrocardiogram. The patient was intubated, mechanically ventilated, and scheduled for urgent coronary angiography as the diagnosis of acute coronary syndrome was established. She was pretreated with 325 mg of acetylsalicylic acid, 600 mg of clopidogrel and 5,000 U of unfractionated heparin. On admission to the cath lab, she remained unconscious with respiratory support, with blood pressure of 90/50 mm Hg, despite the infusion of vasopressors. Physical examination revealed 4/6 systolic murmur with its maximum at the Erb's point, so a transthoracic echocardiography was performed showing akinesia of the inferior wall, hyperkinesia of other left ventricle walls, with ventricular ejection fraction of 75%. Doppler examination revealed a moderate to severe mitral regurgitation and moderate tricuspid insufficiency with the accompanying high-grade pulmonary hypertension (systolic pulmonary artery pressure [SPAP] of 60 mm Hg). After obtaining arterial and venous accesses, an intra-aortic balloon pump (rate 1:1) was inserted and a coronary angiography was performed. Only minor atherosclerotic lesions in the left coronary artery (Fig. 1A) and an occlusion of the left ventricular branch arising from the right coronary artery (Fig. 1B) were noted. Based on clinical status, acute mitral regurgitation due to papillary muscle rupture was suspected. Percutaneous coronary intervention of the occluded infarct-related artery was attempted using Launcher (Medtronic Vascular, USA) 6 French right Judkins 4.0 guiding catheter and soft 0.014-inch BMW (Abbott Vascular, USA) guidewire. The wire could not pass the occlusion site even supported by the balloon catheter. The following wires were used: Whisper MS, Pilot 50, Pilot 150 (all from Abbott Vascular, USA), without success (Fig. 1C). Due to no progress and the prolonged duration of the angioplasty, the procedure was stopped. Suspecting a pulmonary embolism (drop in blood pressure and high SPAP), a pulmonary arterial angiography was performed; this did not reveal any abnormalities. The patient was still unconscious, mechanically ventilated, with the infusion of vasopressors and systolic

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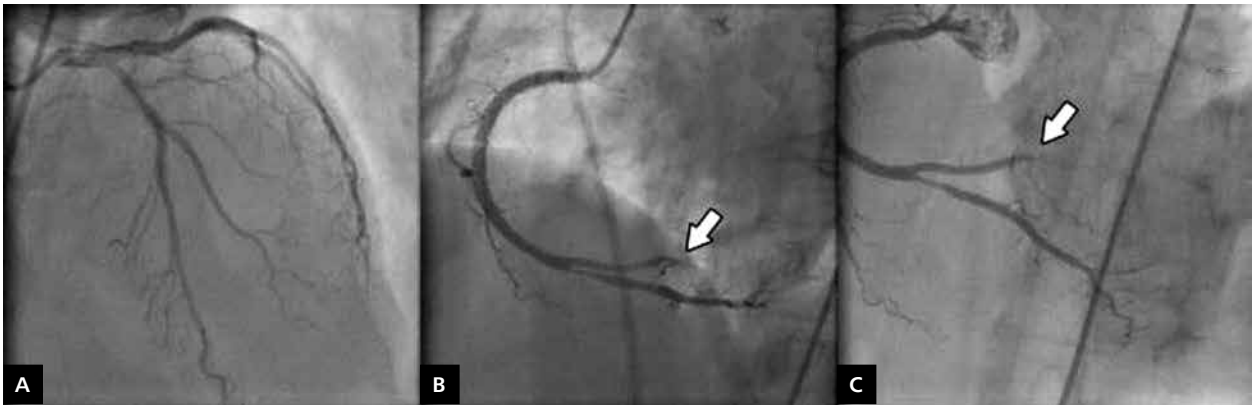

Figure 1. Primary angioplasty in patient with ST-segment elevation myocardial infarction caused by coronary embolisation during the course of infective endocarditis; **A**. Coronary angiography in the anteroposterior cranial view showing mild diffuse disease of the left coronary artery; **B**. Coronary angiography in the left anterior oblique view showing mild diffuse disease of the right coronary artery and an occlusion of the left ventricular branch (arrow); **C**. Final coronary angiography after failed angioplasty of the left ventricular branch (arrow)

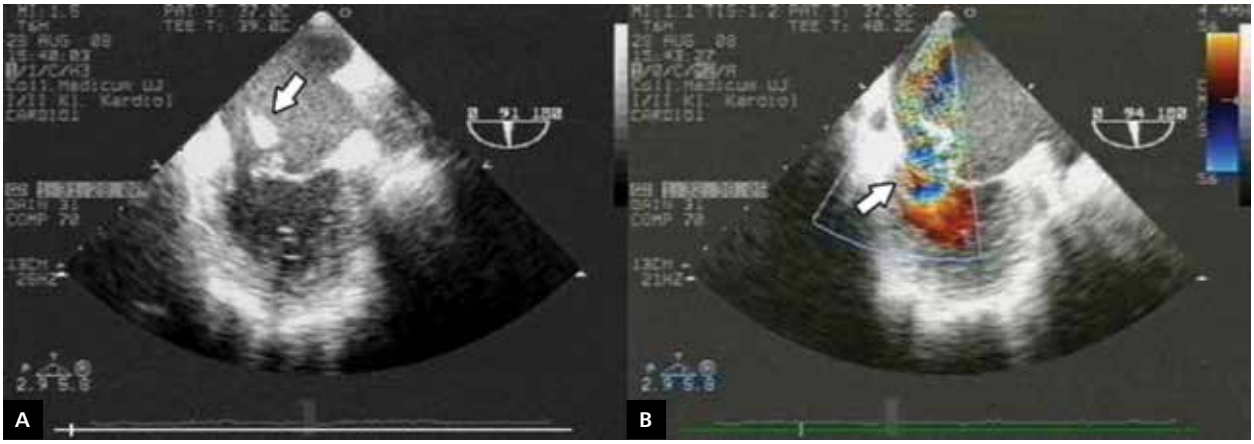

Figure 2. Transoesophageal echocardiogram (TEE) in patient with ST-segment elevation myocardial infarction caused by coronary embolisation during the course of infective endocarditis; **A**. Grey-scale TEE in mid-oesophageal two-chamber view with large vegetation on the posterior mitral leaflet (arrow); **B**. Colour Doppler TEE, mid-oesophageal two-chamber view with severe paravalvular leak (arrow)

blood pressure of about 90 mm Hg. She was transferred to the coronary care unit. Due to a discrepancy between clinical presentations (cardiogenic shock, acute mitral regurgitation) and coronary angiography results (relatively small vessel occlusion) a bedside transoesophageal echocardiography (TEE) was performed to search for a potential, non-ischaemic, cause of mitral regurgitation. TEE revealed massive mobile vegetations on mitral leaflets (Fig. 2A) and perforation of posterior leaflet with severe mitral regurgitation (Fig. 2B). The patient was urgently reported to the cardiac surgery unit, but she was scheduled for conservative treatment. Blood samples were taken for microbiological tests for aerobic and anaerobic bacteria. Empirical antibiotic therapy with ceftriaxone and a reduced dose of metronidazole were started. Peak levels of CK, CK-MB, and troponin I were 1,120 U/L, 94 U/L, and 4.36 μg/L, respectively. Microbiological tests were negative. On the third day after admission, the subject died due to acute heart failure. The presence of vegetation on the mitral valve, perforation of the posterior mitral leaflet, and the left ventricular branch occlusion caused by embolic material from the vegetation was confirmed by autopsy.

STEMI resulting from an infective endocarditis is a rare clinical entity [1, 2]. The left anterior descending artery is the most common site of coronary embolisation by vegetations, originating frequently from the anterior mitral leaflet [3–5]. The second possibility is an occlusion of coronary artery ostium by vegetation arising from the aortic valve. In our case, the left ventricle branch of the right coronary artery was occluded. There is no clear evidence for the treatment of STEMI caused by endocarditis. Anticoagulation does not prevent embolisa-

tion during the course of infective endocarditis, and additionally may raise the bleeding risk. Also, the use of fibrinolytic therapy in STEMI caused by endocarditis may increase the intracranial bleeding risk and may be even contraindicated [3]. Primary angioplasty is thought to be relatively safe, although no data on the safety and efficacy of percutaneous treatment in patients with infective endocarditis is available. Balloon inflation at the site of the occlusion may result in displacement of the vegetation and has relatively poor outcomes regarding vessel patency. A stent implantation might be helpful, but the risk of stent deployment during bacteremia should be taken into account before its use [6, 7]. According to the European Society of Cardiology Guidelines from 2009 [8], infective endocarditis complicated by acute cardiac failure is a major indication for urgent surgery. However, due to STEMI complicated by cardiogenic shock and unclear status of bacteremia at the time of presentation, the subject was scheduled for initial conservative treatment.

This case report serves as a reminder that infective endocarditis can be a rare cause of acute myocardial infarction. Importantly, haemodialysed subjects are especially prone to infective endocarditis. Occluded coronary artery may be treated mechanically, with coronary angioplasty. In a patient in cardiogenic shock and where there is a discrepancy between clinical findings and symptoms, a TEE is worth performing in order to exclude other causes. Such a subject should be also immediately referred to the cardiosurgery department.

Conflict of interest: *none declared*

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